GATING OF PREFRONTAL CORTICAL INPUT TO THE NUCLEUS ACCUMBENS BY THE HIPPOCAMPUS: IMPLICATIONS FOR THE ETIOLOGY OF SCHIZOPHRENIA.

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During *in vivo* intracellular recordings, the neurons of the nucleus accumbens in rats were observed to receive continuous stimulation from excitatory afferents originating in several cortical and subcortical regions, including the glutamatergic afferents from the hippocampus and the prefrontal cortex. Furthermore, the majority of these neurons were found to exhibit a bistable activity pattern, in which the membrane potential showed spontaneous, large-amplitude transitions between steady-state hyperpolarized and depolarized membrane potentials. Stimulation of the prefrontal cortex and the hippocampus evoked monosynaptic membrane depolarizations in all of the cells recorded. Activation of the prefrontal fibers could evoke spike firing only when the neurons were in the depolarized state; this was followed by a rapid transition to the hyperpolarized, nonfiring state. In contrast, stimulation of the hippocampal afferent fibers elicited a prolonged depolarization of the membrane similar to the depolarizing plateaus that occurred spontaneously in the bistable cells. Furthermore, the presence of hippocampal stimulation-induced depolarizations enabled subsequent prefrontal cortical stimulation to evoke accumbens spike discharge, which terminated the depolarizing plateau. Therefore, activation of the hippocampal afferents to accumbens neurons is a prerequisite for prefrontal cortical fibers to activate accumbens cell discharge. This arrangement would thus support a hippocampal-based context dependency for information flow from the cortex to the thalamus. Furthermore, under conditions of hippocampal dysfunction, the absence of hippocampal facilitation of prefrontal cortical throughput should result in a loss of context dependency in addition to a functional hypofrontality in subcortical regions.

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